Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

February 7, 1905.

The President, Dr. Joseph Fraenkel, in the Chair.

Two Cases of Dysbasia Angiosclerotica (Intermittent Claudication), with Demonstration of Calcareous Deposits in the Pedal Arteries.—Dr. J. Ramsay Hunt read this paper, and the demonstration of radiographs was by Dr. Arthur Holding. The first patient was a man, 49 years old. His occupation for the past 32 years had been that of an iceman, which necessitated long and frequent exposure of the feet to cold and wet. The patient had used alcohol and tobacco to excess for many years. He acquired syphilis in 1879. The following year he had some ulcerations on the right leg, and five years later suffered from ulceration of the palate, with perforation. In September, 1903, he had an attack of delirium tremens, for which he was treated in the alcoholic pavilion of Bellevue Hospital. Restraint was necessary, and he was bound in the usual manner by sheets attached to the wrists and ankles. The first symptoms of the present affection appeared immediately after his discharge from the hospital, and was attributed by the patient to the restraint used. He complained of stiffness in the toes and the left leg; also the ankle, and paresthesia of the toes and edges of the feet. After walking a quarter of a mile the prickling, pain and stiffness became so distressing that he had to sit down until it wore off, which was the case after a few minutes' rest. His symptoms were much worse in winter than in summer, and were especially severe on a cold day. On rising in the morning the feet and legs felt perfectly well and natural. Toward the end of the day the feet became puffed up and swollen, and presented a mottled and cyanotic hue. He complained that they felt cold most of the time. The posterior tibial artery was not palpable on either side. The dorsalis pedis was full and larger than normal. The femorals and popliteals pulsated normally on the two sides. The feet were cold, and on standing became red and swollen, with a distinct purplish tinge. Numerous venules and minute venous varicosities were present about both ankles and along the anterior tibial region. The toe-nails on the left foot presented transverse ridges. The reflexes and sensations of the lower extremities were normal. Signs of a general arteriosclerosis were present, and the urine contained a faint trace of albumin, but no casts nor sugar. Its specific gravity was 1.017. Subsequent examination revealed an occasional faint pulsation in both posterior tibials, weaker on the left side.

The second patient presented by Dr. Hunt was a man 60 years old, who for fifteen years had been a longshoreman. For the past ten years he had been exposed a good deal to the cold and wet. He denied lues. For the past fifteen years he had smoked from ten to fifteen cigars daily, and had used alcohol to excess. About five years ago he began to suffer from numbness, stiffness and cold sensation in the toes of the right foot, coming on about two hours after beginning work in the morning. At the end of the first year he was compelled to rest after about three hours. At the end of two years his condition had grown worse; the paresthesia and stiffness had extended as high as the ankle, and rest was necessary after two hours' work. After resting, and in the morning on rising, all his symptoms had disappeared. The symptoms in the left foot gradually grew worse, and six months ago similar symptoms appeared in the right foot, beginning in the toes. Both feet assumed a mottled appearance on standing, but did not swell. A pulsation of the femorals, popliteals and posterior tibials could be felt on both sides, while the pulsation of the dorsalis pedis was entirely absent on both sides, although the artery could be felt as a fine-rounded cord. The reflexes and sensations of the lower extremities were normal. There were well-marked signs of a generalized arteriosclerosis. The urine had a specific gravity of 1.023; it contained a trace of albumin, no sugar. The symptoms presented by the cases shown were to be ascribed to an arteriosclerosis of the vessels of the leg and foot—the so-called dysbasia angiosclerotica (intermittent claudication of Charcot). The X-ray photographs demonstrated by Dr. Holding showed very exquisitely deposits of lime in the posterior and anterior tibial arteries and the dorsalis pedis, marking out very definitely the course of the vessels. As might be inferred from the general evidences of arteriosclerosis in both cases, the pathological process in the pedal arteries was the usual arteriosclerosis of Gull and Sutton, with deposits of lime in the media. These cases are supposed to be less amenable to treatment than the endarteritis forms; hence this method of demonstration might have a certain prognostic value. Both cases had shown a very moderate response to treatment.

Dr. B. Sachs called attention to the similarity of the lesions met with in this condition to those of erythromelalgia.

A Case of Probable Infectious Myelitis of the Cervical Region. Following a Compound Fracture of the Jaw.—This was also presented by **Dr. Hunt.** The patient was a man 36 years old, a painter by occupation, who, until the onset of the present trouble, had enjoyed excellent health. He had never shown any symptoms of lead poisoning. On Dec. 7, 1904, he fell on the street and fractured his lower jaw in two places. The fracture was a compound one, and the wound, which was on the inside of the mouth, bled for several hours. Three weeks later he had rigors at night, on one occasion followed by profuse perspiration. On Jan. 5, 1905, after a heavy slumber, he awoke with severe pain in both arms, which were also very weak, so that he could hardly lift them. The weakness was chiefly in the muscles of the shoulder, especially on the left side. The pains continued all day with great severity, sharp and lancinating in character and shooting through the whole length of the arms, although more severe in the shoulders. The next morning the pains had ceased entirely in the right arm, and the power of the extremity had returned. He was unable, however, to elevate his left arm and the severe pains still continued, especially in the region of the left shoulder. There were no vesical symptoms. For several nights following the onset of the paralysis he had chills of moderate severity. The patient was first seen by Dr. Hunt on Jan. 20, 1905, two weeks after the onset of the symptoms. He still complained of sharp, shooting pains in the region of the left shoulder, and the deltoid muscle was somewhat tender to deep pressure. It might be added, however, that on compressing the muscle bundles of the deltoid between the fingers the same pain and tenderness were elicited as on deep pressure. The nerve plexus above the clavicle and in the axilla and along the inner side of the arm was absolutely free from any tenderness. The left arm was the seat of a slight general atrophy, which was very marked in the deltoid muscle. All movements of the left arm and shoulder could be carried out with practically normal power, excepting adduction of the arm. On attempting adduction some of the posterior bundles of the deltoid were felt to contract, but little or no movement of the extremity was produced. All the arm-jerks were present, and were equal on the two sides. On testing the sensibility it was found that the tactile, pain and temperature senses were retained over both upper extremities, and that the deep sensibility was also normal. A more careful sensory test, however, showed that the sensation was relatively diminished for pain, temperature and touch along the outer side of the arm from the shoulder to the wrist, corresponding very closely to the recognized sensory distribution of the fifth cervical segment of the cord. On the right arm a similar relative obtunding of sensation was also present, but less marked and not so extensive, being practically limited to the region of the deltoid. The gross motor power and tendon reflexes of the lower extremities were normal. The Babinski phenomenon was absent. The deltoid muscle failed to respond at Erb's point to the galvanic current, although good contractions were produced in the biceps and the supinator longus. The direct faradic current produced a sluggish response in the posterior portion of the deltoid; no response in the anterior half. By the direct galvanic current a slow, vermicular response was elicited with reversal of the poles. The urine contained some albumin and hyaline casts. A blood count showed 4,112,000 red blood corpuscles, 10,000 white cells and 75 per cent. of hemoglobin. The erythrocytes were normal; no granular basophilia. The differential count was as follows: polymorphonuclears, 70.6 per cent.; lymphocytes, 10.2 per cent.; large mononuclears, 18.8 per cent.; eosinophiles, 0; mast cells, 0.4 per cent.

On Feb. 1, 1905, the patient still complained of a dull aching, with occasional sharp, shooting pains in the left shoulder. The moderate tenderness in this region still persisted. The gross motor power of the deltoid had greatly improved, so that the arm might now be elevated above the head. This occurred chiefly through the medium of the posterior half of the deltoid muscle, the anterior portion remaining entirely relaxed and flaccid. The slight sensory changes still persisted. There was no Babinski phenomenon. A small piece of dead bone had been removed from the inside of the mouth. The breath and excretions from the mouth were very fetid. The interesting features of this case, Dr. Hunt said, were those of localization and etiology. Considering the patient's previous good health and the absence of any other demonstrable cause, it was natural to refer the symptoms to the suppuration and the carious process in the lower jaw, more especially as mild symptoms of sepsis directly preceded and followed the onset. While the persistent pains and apparent tenderness over the left deltoid might suggest a neuritis of the circumflex nerve, it might be emphasized that the paralysis of the deltoid was incomplete, and evidences of objective sensory changes limited to the circumflex were absent. The speaker also emphasized the absence of tenderness or other evidences of inflammation in the plexuses and cellular structures of the neck. On the other hand, the initial paralysis and severe pains, symmetrical and bilateral in distribution, the rather sudden recovery of the right arm and the more gradual recovery of the left; the unequal distribution of the paralysis in the left deltoid, and the gradual improvement which this paralysis had undergone; the relative diminution of sensation on both sides in an area corresponding very closely to that of the fifth cervical segment, all pointed strongly to a central cord lesion. The nature of this case could only be inferred. A poliomyelitis or a myelitis of infectious origin naturally suggested themselves.

Dr. Charles L. Dana said he had seen Dr. Hunt's patient some days ago, when there was still some paralysis of the left arm. The condition could best be explained on the theory of a thrombosis, plugging one of the central arteries, and affecting the left anterior horn, about the region of the fifth cervical segment.

Dr. Fraenkel said there were a number of cases reported in literature where septic conditions, particularly that known as angina Ludovici —an acute suppurative condition of the connective tissues of the neck— had given rise to a septic neuritis. The speaker said he saw such a case in consultation about three years ago. The case was one of a septic throat condition, and eight or ten days later, after the acute symptoms had subsided, there were evidences of a bilateral brachial neuritis. It began with severe pain, in that way differing from the case shown by Dr. Hunt.

Address of the Retiring President, Dr. Pearce Bailey.—The speaker stated that while the membership of the Society had been materially enlarged during the past year, notably by younger men, the new members had not shown the active participation in the proceedings which was absolutely essential for the Society's continued prosperity. Many interesting cases had been shown during the year, and the general discussions had brought out large audiences, and had been fruitful in valuable information. While individual clinical cases and general discussions perfected the art of the clinician, they did not give neurology all that neurology had a right to expect from as representative a body as the New York Neurological Society. For the science to be advanced, more fundamental contributions were essential. It was commonly said, and with much truth, that neurology was living to-day on its past achievements. So much the more necessary was it to try to give it new life, to start it again on that progress which, only a few years ago, made it the most brilliant branch of medicine.

Address of the President-elect, Dr. Joseph Fraenkel.—The speaker stated that while neurology had a glorious past, its future, viewed with the bias of the sentiment prevailing at present, appeared discouraging. A pessimistic attitude had crept in; the transactions of the meetings of neurologists seemed to lack the animation and enthusiasm of former days. Why this was so he did not know; it had happened because it had to—it was a phase of evolution. Dr. Fraenkel said that personally he did not believe that the present augured ill for the future. There was a wealth of fundamental questions awaiting investigation. New methods had been elaborated that promised valuable aid. Chemopathology, which seemed especially adapted for the elucidation of some neurological questions, with its promise of better therapeutics, was well established. The physiology of the ductless glands was closely allied to neurological themes and deserved particular attention. All neurologists were thoroughly dissatisfied with our conception and interpretation of the so-called functional conditions. It was encouraging to note a decidedly rational spirit in the newer literature on the subject. The relation of the nervous system to the pathology of the vegetative functions was a subject which the speaker believed to be of fundamental importance to clinical neurology and psychiatry. The subject had not been lacking in votaries, but had proven refractory and unyielding. A good deal of preparatory work had been done for the better understanding of the subject. So-called visceral or vegetative phenomena accompanied more or less prominently organic disease of the nervous system, and constituted the chief physiognomy of all functional states and functional symptom-complexes. Trophic, vasomotor and secretory disturbances of the skin, changes in the trophism of joints and viscera, disturbances of the physiologic rhythm of the vaso- and visceromotor innervation, perversions of visceral sensation, were often seen at the bedside, and were not sufficiently appreciated. The visceral and vegetative symptoms of tabes and hysteria were good illustrations of familiar types. The innervation of the ductless glands was manifestly a matter of importance, and the histology of the blood-vessels of the brain and their innervation were morphological questions of broad application to physiology and pathology. Phenomena of disordered trophic influence were not uncommonly encountered by the neurologists, and very little was known about most of them. The palmar and plantar *mal perforans* of tabes, the cutaneous ulcerations of syringomyelia, the decubital ulcers observed in the course of organic disease of the cord or brain, the hysterical skin eruptions, the glossy skin, scleroderma, morphea, etc., were hardly understood. Atrophy of the hair of the limbs in tabetics, and hypertrichiasis in cases of poliomyelitis were frequently seen. It was interesting to observe that in cases of disease of the anterior horns, when the disease was limited to the anterior horn cells, as in amyotrophic lateral sclerosis, for instance, this change in the growth of the hair did not take place. Atrophy of muscles severed from their connection with the peripheral nerves was frequently observed. Atrophy of visceral muscles under similar conditions was doubted. The trophic changes of the joints in tabes and syringomyelia, and the nature of arthritis deformans were still mysteries. The pituitary body was generally believed to exert trophic influence upon some parts of the skeleton. A large number of vasomotor disturbances were still puzzling problems. The general vasomotor ataxias observed so frequently in the course of Graves' disease and the climacteric neuroses were, on clinical grounds, referred to perverted function of the thyroid glands or of the ovaries, respectively. Angioneurotic edema was believed by many to be explained on the basis of a toxemia. The question of erythromelalgia was still *sub judice*. Sachs and Wiener's findings of disease of the bloodvessels and peripheral nerves had been frequently corroborated. The condition was in danger of losing its clinical entity and of being classed with the arterial diseases of the extremities (angina pectoris of the foot, intermittent claudication). For explanation of the spasmodic disturbances of the respiratory function, asthma nervosum, tachypnea, hysteric dyspnea, little definite pathologic information was at hand. The bradycardia of direct and indirect vagal lesions was frequently described. According to Oppenheim, neuritis of the vagus was indicated by acceleration, rarely by slowing of the heart beat. The vast variety of symptoms referable to disordered innervation of motion, sensation and secretion of the gastro-intestinal tract were well known and often very perplexing. Cases of cord lesions were not infrequently met with which began with visceral, particularly gastro-intestinal symptoms, and which were often treated for a long time under this mistaken diagnosis. In a large number of cases of lead colic the celiac nerves and ganglia were found evidently diseased. Changes in the constitution of the urine were frequently observed in nervous diseases. Albuminuria was often a symptom of a pontine lesion, while glycosuria was frequently associated with disease of the posterior fossa. The symptomatology of disordered genital functions was also beginning to be studied along lines suggested by Dr. Fraenkel.

March 7, 1905.

The President, Dr. Joseph Fraenkel, M.D., in the Chair.

A Case for Diagnosis.—Dr. Smith Ely Jelliffe presented this case for diagnosis. The patient was a woman, 33 years old, a dressmaker by occupation. She had seven sisters and one brother alive and in good health. One sister had died of consumption. Her father, who was alive at 65, was a hemiplegic. Her mother was 70 years old, and enjoying good health. The patient had scarlet fever at the age of 7 years. Following this attack, she was weak for a long time and would fall easily. There was no history of hemiplegia at this time. Later in childhood she had measles, whooping cough and diphtheria. She had an attack of jaundice when she was 22 years old, and malaria six years ago. Her menstruation began when she was about 14, and was regular, lasting two days. Some years ago she had an attack of sciatica, and gave a history of painful swellings in the axillæ, elbows and knees, probably due to some lymphatic infection. When she was 18 she fell, striking the back of her head, resulting for a time in severe headaches.

Her present illness began two years ago, and its onset was gradual.

She first felt pain in the little finger of the left hand. This was burning in character, and ran up and into the shoulder along the course of the sterno-cleido-mastoid to behind the ear. The pains were persistent and grew more general. Then the hand began to shake and grow weaker. The pain had lasted two weeks before the tremor and weakness became apparent. The tremor at first was very pronounced. About a year later she noticed a loss of power in the left leg, with stiffness and drawing up of the muscles and some pain in the knee. During the past summer the pain had been referred to the ankle. She also complained of frequent attacks of diarrhea, with colic. There were no bladder symptoms. An examination of the eyes showed slight transitory diplopia, but no permanent nystagmus. No impairment of sight. There was slight transitory inequality of the pupils; no Argyll-Robertson pupil. Consensual light reflex normal. Skin reflexes normal, No pains; no anesthesia; no disturbance of heat and cold sensations; no Romberg. Slight tremor of head at times. Musculature good. There was weakness and stiffness of the entire left side, more marked in the upper than in the lower extremity. No sensory changes in the entire body. The reflexes were increased on the left side. No Babinski; no clonus; no tremor of leg. The tremor of the arm was coarse, of the semi-intentional type, but not typical. The heart sounds were exaggerated, but there was no murmur. The leg dragged; there was marked stiffness and rigidity. No electrical changes; no atrophy. The patient said that her memory was not as good as formerly, especially for recent events. Otherwise no change was noted. Her friends stated that she was more irritable than formerly, but this was perhaps natural, as the tremor of the hand prevented her from carrying on her business as a dressmaker. The speaker said the symptoms were suggestive of a multiple sclerosis, a spinal tumor, or a progressive hemiplegia of unknown causation.

Dr. L. Pierce Clark, who had also seen the patient at the Vanderbilt Clinic, said the case impressed him as one of multiple sclerosis, slowly progressive, and probably of the cerebral type. There were some evidences of involvement of the spinal cord.

A Case of Myotonia Congenita.—Presented by Dr. Graeme M. Hammond. The patient was a girl of 16, the youngest of three children. The other children were boys. One of them, aged 18, died suddenly a week ago of heart disease. The child's parents were cousins. There was no other case of myotonia on either side of the family for three generations; beyond that nothing was known of the ancestors.

The patient began to walk when she was ten months old. With the exception of measles in childhood, she had always been well and healthy. When she was about two years old the mother noticed that the child, after sitting awhile, had great difficulty in getting up and walking. She arose slowly, on account of great stiffness in the muscles, and often fell. On attempting to walk she at first found it impossible to move, so rigid were her legs. Finally she would move one leg stiffly, then the other, and began to walk with stiff, mechanical movements, like "a nursery doll." The more she walked the more limber she became, and in a few minutes walking became easy and natural, and she could even run and dance. This condition had persisted all her life.

She menstruated for the first time in March, 1904; then not again until September, 1904, and since that time she has not menstruated at all. Her body and arms were rather small, but the legs and thighs were very large. The knee-jerks, after sitting awhile, were present and responded quickly, but the movement was short. After walking, when the legs were quite limber, the knee-jerks were normal. Both calves measured 14¼ inches; the thighs (7 inches above the patella) 20¼ inches. The legs were 33 inches long. The muscles of the arm reacted normally to both forms of the electrical current. The legs seemed to respond normally to faradism. When galvanism was used the muscles responded sharply and deeply to make of current, then immediately relaxed, then a second contraction ensued, which was not so well marked as at first. The muscle, when it contracted, did so with a wave-like or vermicular movement. The same muscular tonus was observed after standing or lying down. The most difficult time the patient had was on attempting to rise in the morning.

Dr. G. L. Walton, of Boston, spoke of the liability of these cases to be mistaken for hysteria, on account of the apparently meaningless transition from clumsiness to agility. Continuous observation of the patients showed an unvarying adherence to fixed rules referable to disturbance of the neuro-muscular mechanism, and independent of the mind. Dr. Walton also referred to a symptom that was present in two typical cases of myotonia congenita that had been under his observation, namely, the slow and clumsy ascent of stairs, followed by an active and nimble descent. This trial was then made upon Dr. Hammond's patient, with the same result.

A Case of Progressive Muscular Atrophy of the Left Upper Extremities.—Presented by Dr. Edward D. Fisher. The patient was a young woman, whose occupation was that of a machine worker, passing articles along under a machine, the left hand being engaged more than the right. Her family history was negative as was also her past history, with the exception of the fact that when she was about ten years old she complained of pain in the upper arms, between the shoulder and elbow, when carrying her school books.

About two years ago she began to notice some weakness in the left hand, together with some tendency to stiffness, especially when playing the piano. This was followed by wasting of the muscles, which appeared to be typical of progressive muscular atrophy. One peculiarity of the case was the extreme icy coldness of the hands, in spite of efforts to keep warm. The electrical reactions were impaired. There were no sensory disturbances; no symptoms pointing to syringomyelia; no scoliosis. The atrophy was gradually extending up the forearm.

Dr. William M. Leszynsky said the case reminded him of one he saw recently in which there were various symptoms affecting the upper extremity, and in which the presence of a cervical rib was subsequently demonstrated. This abnormality, the speaker thought, should always be looked for with the X-ray in doubtful cases of this character, and wauld, perhaps, often explain symptoms which would otherwise be attributed to some lesion of the brachial plexus.

Dr. L. Pierce Clark said that he had recently seen two cases of early progressive muscular atrophy in which the symptom observed in Dr. Fisher's case, that of coldness of the skin, was present. However, the symptom was confined quite distinctly to the skin distribution of the ulnar nerve in one case, and in the other the coldness occurred in the entire skin area of the first dorsal segment of the cord. He believed, in these cases at least, that progressive muscular atrophy was a lesion of the peripheral nerves as well as of the anterior horn of the cord, consequently the sympathetic nerves were damaged, as in traumatic peripheral neuritis. Recent autopsy studies on progressive muscular atrophy would seem to confirm this pathological explanation.

Dr. William G. Spiller of Philadelphia said that in his experience progressive muscular atrophy of spinal origin was one of the rarest of all nervous diseases. He recalled one case, reported by Dr. C. S. Potts from the University of Pennsylvania's nervous clinic, in which the atrophy began in the extensors of the forearm. The explanation of the condition in that case was that the man's occupation was that of a fireman, and the atrophy began in the muscles that were most constantly used.

Two Cases of Nerve Anastomosis.—Presented by Dr. Alfred S. Taylor. The first patient was a boy, 8 years old, who three years previously developed an attack of anterior poliomyelitis which involved the right hand and forearm. The hand was entirely useless, but as the muscles of the shoulder and upper arm were in fairly good condition, the case was regarded as a suitable one for nerve anastomosis.. Accordingly, a few days ago the brachial plexus was exposed and the 7th and 8th cervical and 1st dorsal roots were divided, turned up and inserted by lateral anastomosis into the junction of the 5th and 6th nerves. The anastomosis was effected without any tension on the nerves. The wound was then closed and the parts immobilized. The dressings were still in place and the ultimate outcome of the operation, so far as improvement of function was concerned, was doubtful.

The second patient, shown by Dr. Taylor, was a boy, about 8 years old, who in the early summer of 1903 had scarlet fever, followed by otitis media and mastoiditis, for which he was operated on July 13, 1903. at the Randall's Island Hospital. Three days later a facial palsy appeared, and rapidly progressed to complete paralysis. On October 24, 1903, three and one-half months later, complete R. D. was found on electrical examination. A facio-hypoglossal anastomosis was immdeiately done under chloroform anesthesia. The facial nerve was found to be soft and friable, and gray in appearance. But one suture could be made to hold to the hypoglossal, which in turn was under such tension that a fine suture was passed through the hypoglossal sheath to anchor the nerve leading to the neighboring fascia, and so prevent tension on one anastomosis, as far as possible. Primary union was obtained. There was almost complete palsy of the muscles supplied by the hypoglossal immediately after the operation. Ten weeks after the operation some difficulty of speech remained, owing to imperfect use of the tongue. Difficulty in swallowing and mastication, and unilateral furring of the tongue, all of which were present for some weeks after the operation, and which were due to operative injury of the 12th nerve, disappeared. Three months after the operation the face was far less assymetrical than before, and the eye could be partly closed. R. D. was unchanged. At the end of eight months there was slight motion about the right side of the mouth. The improvement slowly continued and now, fourteen months after the operation, the patient had a fair amount of control over the formerly paralyzed side of the face.

Dr. B. Sachs thought that three and one-half months was rather too soon to consider an operation in a case of facial palsy, as in many of these patients improvement began after a much longer period than that which had elapsed.

Dr. Hammond said that he did not think the return of power in the paralyzed muscles in the case shown by Dr. Taylor proved that the anastomosis had been successful. He recalled the case of a young lad who developed a facial palsy after a mastoid operation, and without any attempt at anastomosis, motion in the affected muscles was first noticed at the end of fifteen months, and eventually was absolutely restored.

Dr. Leszynsky said he had seen a great many cases of Bell's palsy following mastoid operations, and in his experience a favorable outcome, like that reported by Dr. Hammond, was rather unusual. He had followed some of his cases for a long time, and had failed to note any improvement.

Further Report of a case of Hypoglossal Facial Anastomosis.—By Dr. Charles H. Frazier, of Philadelphia. The speaker said that his first opportunity to operate upon a case of facial palsy was in August, 1903, when he saw, in consultation with Dr. William G. Spiller, a patient who, five months before, in a fit of mental aberration had shot himself with a revolver, the ball entering the external auditory meatus. This resulted in a complete facial palsy, and from the manner in which the wound was inflicted and the point of entrance of the bullet, there was every reason to believe that the facial nerve was completely severed in its passage through the petrous portion of the temporal bone before it made its exit from the stylomastoid foramen. The case was one eminently suitable for operative intervention, as the nerve was irreparably damaged and the muscles hopelessly paralyzed. The operation consisted in a hypoglossal facial anastomosis. During the eighteen months that had elapsed since the operation, which was done in September, 1903, the patient had been examined from time to time, and there had been a slow, but progressive improvement.

One month after the operation there was no response to the faradic current in any of the muscles. Reaction of degeneration and polar changes were marked. Six months after the operation there was no change in the condition. Nine months after the operation there was still no response to the faradic current, while with the galvanic current there was lessened irritability. At this date there appeared for the first time a peculiar involuntary movement of the angle of the mouth. Fourteen months after the operation there was still no response to the faradic current, but a slight increase of irritability under the galvanic current. Polar change still distinct. Seventeen months after the operation voluntary movements at the angle of the mouth were for the first time observed. In addition to this the patient was able to almost completely close the eye. All the muscles responded to the faradic current.

Dr. Frazier called attention to the fact that it was not until after the fourteenth month had passed that a positive reaction to faradic stimulation was observed, with one exception, namely, the orbicularis palpebrarum, which responded slightly in the ninth month. Another interesting point was the restoration of an involuntary before a voluntary movement. In those cases in which the anastomosis had been made with a branch of the trunk of the spinal accessory nerve, associated involuntary movements were the rule rather than the exception. The speaker called attention to the order in which the muscles regained their power, beginning with the muscles of the lower part of the face, those supplied by the branches of the cervico-facial division, and extending gradually to the upper part of the face, or those supplied by the tempero-facial division.

One of the arguments that had been advanced against the selection of the hypoglossal nerve was the resulting atrophy of the tongue. Section of the hypoglossal nerve would be followed by paralysis of the depressors and some of the elevators of the hyoid bone, with atrophy of one-half the tongue. The speaker said that apart from the misshapen appearance of the tongue, which was not to be compared in importance with the facial deformity one was striving to relieve, he had not found any other disturbance arising from section of the hypoglossal nerve. In none of his cases was there difficulty in swallowing or talking.

Dr. Frazier said he thought no one would question the propriety of the operative treatment of facial palsy. The operation was founded upon a psychological law, proven by experimentation, and the clinical experience of a number of observers had established all that was claimed for it. The only feature in the technique of the operation worthy of discussion was the choice between the eleventh and twelfth nerves. In a previous communication on the subject (University of Pennsylvania Medical Bulletin, November, 1903) the speaker said he expressed a preference for the twelfth nerve, and from subsequent investigation of the results of other operators and from his own experience, he still held to the same opinion. The whole question hinged upon the importance which was attached to the associated movements. So far as he had been able to learn from the recorded cases of spinal-accessory-facial anastomosis, there had not been a single instance in which a voluntary effort to raise the shoulder had not been attended with contraction of the facial muscles, and these so-called associated movements constituted a very serious objection to the selection of the eleventh nerve. As to the method of making the anastomosis, theoretically, an end-to-end anastomosis should secure the best results.

Some Newer Ideas on Nerve Anastomosis.—By Drs. William G. Spiller and Charles H. Frazier.

A Contribution to the Pathology and Surgical Treatment of Chronic Facial Palsy. By Drs. L. Pierce Clark, Alfred S. Taylor and Thomas P. Prout. (Read by Dr. Clark.) The reader said it was not known why the facial nerve suffered from palsy with such frequency, nor was there as yet adequate information regarding the nerve elements or the part of the nerve that was primarily involved. The tendency of Bell's palsy was probably due to anatomic causes. The nerve had a tortuous and exposed course through the Fallopian canal, and the extension of inflammation from the middle ear, or the effect of cold acting through this channel upon the facial nerve, was easily understood. Facial palsy, both in man and animals, was relatively infrequent when the face and head were properly protected. It seemed reasonable to suppose, also, that a certain number of individuals suffered from a congenital narrowing of the foraminal exit of the nerve.

There were good reasons to believe that the initial changes of this condition took place in the perineural sheath, although the exact pathogenesis of Bell's palsy was still uncertain, because of the meagerness of the material at hand for study. In two cases of typical, uncomplicated Bell's palsy coming under the author's observation and treated by facio-hypoglossal anastomosis, a section of the affected nerve was excised and submitted to Dr. Prout for microscopical study. In the first of these cases the paralysis had existed for three and one-half months; in the second, for twelve years. Dr. Prout reported that in the first case the nerve was in a condition of incomplete degeneration. There were still a few nerve fibers undegenerated, or in which the myelin sheath was only partially broken up, and these stood out in marked contrast to the degenerated nerve fibers in which they were imbedded, which latter showed here and there fragments of myelin. There was, furthermore, a marked invasion of the tissues with leucocytes, and the cells of the neurilemma were shrunken and shrivelled in some portions; in others the nuclei of the neurilemma cells were large and well-defined. The perineural sheath showed marked infiltration with leucocytes.

In the second case, which was of a chronic character, the nerve fibers were completely degenerated, showing but occasional granules belonging to the myelin sheath. There were no undegenerated fibers. The tissues belonging to the perineural sheath showed the most decided changes. There was marked proliferation of the cells belonging to the perineural sheath, with infiltration of the tissues, and clumps of cells scattered here and there throughout the section. This tissue exceeded the proper nerve tissue of the section by about two to one. The neurilemma cells were markedly shrunken and shrivelled.

The painlessness of Bell's palsy even to external pressure on the nerve trunk and the paralyzed muscles argued for a simple degenerative neuritis in the peripheral portion, and not a parenchymatous affection. There was good reason to believe that Bell's palsy was primarily a Fallopian neuritis, which through strangulation of the nerve strands induced a low grade of secondary degenerative neuritis in the periphery.

Dr. Alfred S. Taylor said that his technique of facio-hypoglossal nerve anastomosis was described in detail in a paper upon the surgical treatment of facial palsy by Dr. L. Pierce Clark and himself, which appeared in the *N. Y. Medical Record,* February 27, 1904. The operation involved the following steps: (1) The incision. (2) Isolation and section of the facial nerve. (3) Exposure of the hypoglossal nerve. (4) The implantation. (5) The closure of the wound. (5) The after-treatment.

The incision, involving the skin and subcutaneous tissue, passed along the anterior margin of the mastoid process and the sterno-mastoid muscle for about 5 cm., starting at the level of the external meatus. When the facial nerve was identified, it was enucleated from the surrounding connective tissue, and divided as far up in the stylo-mastoid forearm as a narrow-bladed knife would allow. Usually one could get from one to two cm. of free nerve trunk.

The isolation of the hypoglossal nerve was the most difficult and tedious step, and involved whatever danger there was in the operation. Once identified, it was dissected upward until the stump of the facial nerve could be approximated to it without tension. This had to be done with care, so as not to divide the minute branches from the pneumogastric, the upper ganglion of the sympathetic and the two upper cervical branches, all of which were in the immediate neighborhood. While the nerve was supported on a blunt hook, a slit one-half cm. long was made well into the nerve trunk. A fine curved needle was threaded to one of each pair of long silk ends previously left tied to the stump of the facial nerve. One suture was passed through the inner and the other through the outer margin of the wound in the hypoglossal nerve. When the sutures were tied the wedge-shaped end of the facial was snugly held in the cleft of the hypoglossal nerve, and was usually best turned slightly upward by means of a probe, a procedure suggested by Dr. Weir. These sutures should not be tied too tightly, lest they injure the fibers of the hypoglossal nerve, a few of which were almost surely included in their grasp.

To prevent the ingrowth of connective tissue elements, Cargile membrane was wrapped about the nerve junction.. The hypoglossal was then dropped back to its normal position, and there was usually no tension on the tissues.

The after-treatment was a most important feature in obtaining the desired result. Massage, electricity, and later coördinate muscular movements, should be persistently and systematically resorted to for months.

As to the time when the operation should be done, Dr. Taylor said that that must be left to the neurologist. The muscles should be kept in good condition. It was known that there was a return of motor power in some instances even after many years had elapsed.

In connection with his remarks Dr. Taylor reported six cases of hypoglossal nerve anastomosis, and showed a number of new instruments that he had designed for the purpose of rendering the operation easier technically, and of avoiding distressing sequelæ. These instruments were illustrated and described in detail by Dr. Taylor in the *N. Y. Medical Record,* March 4, 1905, page 359.

Dr. Charles A. Elsberg reported the following case of facial-spinal-accessory anastomosis. This report, he said, must be regarded as preliminary, as the time that had elapsed since the operation was not long enough for a complete report.

C. F., female, 30 years old, was referred to Dr. Elsberg in May, 1904, by Dr. H. Heiman. When the patient was six months old she had an attack of convulsions, followed by paralysis of the left side of the face, with loss of smell and hearing on the same side. In spite of treatment, the paralysis did not improve. Massage, electricity, injections of strychnine, etc., were continued for many years without any improvement.

When Dr. Elsberg first saw the patient she presented the characteristic features of an old facial palsy. Dr. B. Sachs was asked to see the case in consultation, and found that the left facial paralysis was almost complete. There was a slight faradic response to the lowermost branch of the nerve and the galvanic response was diminished, but the polar formula was not altered in the orbicularis oris or in the nerve itself. There was partial lagophthalmus. Examination of the ear and nose was negative.

After careful consideration it was decided that, in spite of the long duration of the paralysis, an operation might be attended with success. This was done on May 10, 1904, about nine and a half months ago. The facial nerve was anastomosed with the spinal accessory by slitting both nerves and joining the branch of the 11th nerve to the trapezius muscle to the separated portion of the facial with fine silk sutures. Thus neither the 11th nor 12th nerves were entirely divided. The wound healed by primary union, and the patient was discharged from the hospital in two weeks.

Briefly, the after-course was as follows: There was complete paralysis of the trapezius and a partial paralysis of the left sterno-mastoid after the operation. This disappeared in about six months. Eighteen days after the operation the patient declared she had a continual feeling of tenseness in the left side of the face. On the 26th day she declared that she could close her eyes better, and that the left eye did not tear any more. On the 96th day the patient wrote from the country that she had noticed that she was able to move the left corner of her mouth a little, and that she could close the left eye better.

On the 122d day the orbicularisoris contracted strongly to the faradic current; the muscles of the lower lip slightly to galvanism. The same muscles contracted when the spinal accessory was stimulated. Associated movements of the left shoulder and the left side of the face could never be obtained. On the 166th day the patient was able to draw the left angle of her mouth slightly upward and outward, and could close her left eye almost completely. From that time up to the present there had been a slow, but steady, improvement, additional muscles beginning to contract to galvanism and faradism every few weeks.

The case was unusual on account of the duration of the paralysis at the time of operation (29½ years), and because of the absence of associated shoulder movements.

Dr. B. Sachs said that in deciding the question whether to anastomose the facial nerve with the hypoglossal or the spinal accessory his preference would be in favor of the latter procedure, as it involved a nerve of lesser dignity than the former. The hypoglossal was a nerve of great importance, and if the same result could be obtained by substituting the spinal accessory he would certainly favor it. The entire question of nerve transplantation was an extremely important one, and the physiological aspects of the problem were still unsolved. The speaker said he sincerely hoped that the procedure would prove beneficial in hemiplegia, but until further experimental work had been done that aspect of the subject could not be intelligently discussed. In facial palsy several cases had been reported where the nerve anastomosis had been done three and a half or four months after the onset of the disability. This, he thought, was too early, as many cases were on record where improvement began much later without treatment or in spite of treatment. A year at least should be allowed to elapse before operation.

Dr. Leszynsky said he was interested in the remark made by Dr. Frazier that the power would be restored with the return of the faradic irritability. The speaker said he had seen patients with facial paralysis of long standing in whom the faradic irritability had returned although there was no return of motility. These were exceptional cases, but they had been observed, so that the mere return of the faradic irritability did not necessarily indicate that there would be a return of motility. The speaker said he agreed with Dr. Sachs that three or four months was too soon to operate on these cases.

Dr. Walton said that Dr. Spiller's proposition should have the benefit of trial. In disease or injury of the lower neurones every gain in motion, however slight, was gladly welcomed, and if hemiplegics could be helped by nerve transplantation, there was the added hope of lessening the cerebral spasm to which Dr. Spiller had referred, and which in one case coming under the speaker's observation was so troublesome that the arm was amputated at the shoulder at the request of the patient. The brilliant result in Dr. Elsberg's case demonstrated the possibility of transplanting the spinal accessory without causing associated movements, which seemed to be the chief drawback to shoosing this nerve in preference to the hypoglossal.

Dr. Spiller, in closing, said the statement made by Dr. Taylor that in late cases, where the paralysis had existed for a long time, the nerve may have disappeared, was correct. The speaker recalled such a case where the facial nerve could not be found at all. In facial palsy one of the earliest evidences of a return of power that could be elicited was a slight deepening of the naso-labial fold when the eyes were closed tightly.

Dr. Frazier, in closing, said that in his cases of nerve anastomosis the hypoglossal had been selected in preference to the spinal accessory purely on theoretical grounds and a study of former operations, and also because associated movements had followed the selection of the spinal accessory. The case reported by Dr. Elsberg was the first one in which these associated movements had not occurred. Henceforth, Dr. Frazier said, he would favor the end-to-side amastomosis in preference to the end-to-end method.

Dr. Clark, in closing, said he thought the proper time to operate could be fairly accurately decided upon by the results of the electrical tests, and he did not think it wise to fix an arbitrary period of time. In most forms of peripheral neuritis the electrical tests formed a basis of prognosis that was almost absolute. Certainly, at the end of three or four months, if the electrical changes should complete degeneration of the nerve and there was no evidence of repair, he thought an operation would be indicated. At the same time, if one preferred to wait a year, or even thirty years, he saw no objection to it.

Dr. Taylor said the disappearance of tearing to which Dr. Elsberg referred in his case had also been noted in one of his cases. The operative damage to the hypoglossal and the tongue symptoms resulting therefrom were only temporary, and he did not think they should be looked upon as a serious objection to the selection of that nerve for the anastomosis.

PHILADELPHIA NEUROLOGICAL SOCIETY.

March 28, 1905.

The President, Dr. Joseph Sailer, in the Chair.

A Case with Some of the Symptoms of Multiple Sclerosis Due to Trauma.—This paper was presented by Dr. Ralph Pemberton.. On the 16th of January, 1905, the man fell from the upper girders of the Subway, where he was working, a distance of about 16 feet, though whether he stepped into some opening or lost his balance while at work he cannot say. He became unconscious and does not know how he struck, though he fell on a cement flooring. He was taken to the Medico-Chirurgical Hospital and was treated there for four weeks, of which time he can give no satisfactory account, though he was not constantly in bed, and was told that at times he had to be catheterized. On two occasions plaster jackets were applied, the second of which alone he remembers. He